PARENTAL SMOKING AND COMPROMISED CHILDREN

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COMPROMISED CHILDREN

The literature on environmental tobacco smoke includes a body of research on asthma and the relationship between parental smoking and preexistent disease in children. While only a few studies have been conducted thus far on children with, for example, cystic fibrosis (a genetic disorder whereby lung passages often become blocked by abnormally behaving mucus), there have been numerous studies that have examined asthmatic children and the presence of smokers in the home. The results of the studies have been variable and are subject to the influences of many different confounders such as socioeconomic status, genetic determinants, damp housing, and gas cooking in the home. It is not suprising, therefore, that researchers are inconsistent in their interpretation of the available data on exposure to ETS and childhood asthma. Following is a presentation of the major studies that have examined a possible association of ETS with asthma in children. Also included are the studies available on children with cystic fibrosis.

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RESULTS OF SELECTED STUDIES: COMPROMISED CHILDREN

Leeder, et al., 1976	Reported that episodes of asthma in the first five years of life showed an association with parental history of asthma-wheeze and that there is little relationship between asthma in the first five years of life and other family, social, or environmental factors.
Fergusson, et al., 1985	Reported that maternal smoking increased the risk of lower respiratory infections/symptoms during the child's first two years of life, and that after two years this association seemed to disappear. Also reported that there was no increased risk of asthma or asthma attacks attributable to maternal smoking.
Horwood, et al., 1985	Reported that there was no evidence to suggest that the structure, practices (including parental smoking), or the dynamics of the family played a significant role in the development of childhood asthma.
Murray, et al., 1986	Reported that maternal smoking aggravated symptoms in asthmatic children.

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Anderson, et al., 1987	Reported that sex of child, mother's age at the child's birth, pneumonia, whooping cough, tonsillectomy, adenoidectomy, allergic rhinitis, eczema and periodic abdominal pain/vomiting attacks were associated with the development of childhood asthma.
Evans, et al., 1987	Reported that parental smoking increased the number of emergency room visits of children with asthma. The lack of an association between parental smoking and symptoms in asthmatic children caused the authors to question the mechanism whereby parental smoking would increase ER visits.
Toyoshima, et al., 1987	Reported that it was not clear whether parental smoking increased the incidence of asthmatic disease in children.
Kershaw, 1987	The authors conceded that the association observed with parental smoking may reflect a relationship of smoking behaviour to a number of other social factors such as medical care utilization and maternal stress.
Murray, et al., 1988	Reported that paternal smoking, including the number of cigarettes the father smoked at home, had no association with any test results. Maternal smoking in the "wet and cold" season was reported to increase the severity of the child's asthma. There was no assocation in the "warm and dry" season

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	and the severity of the child's asthma. The authors attribute this to increased ventilation in the "warm and dry" season.
Somerville, et al., 1988	Reported a positive association between parental smoking and asthma in girls and a non-significant negative relation between parental smoking and asthma in boys.
Oldigs, et al., 1990	Reported that in children with mild bronchial asthma one hour of passive smoke exposure did not cause airway obstruction or changes in bronchial responsiveness.
Sherman, et al., 1990	Neither bronchiolitis, eczema, croup, personal cigarette smoking, maternal smoking, paternal smoking, nor delivery complications bore an apparent relation to the development of childhood asthma.
Weitzman, et al., 1990	The authors concede that the estimate of children's exposure to cigarette smoke is "crude", based on parent reporting of smoking during pregnancy.
Rubin, 1990	Reported an association between parental smoking and the severity of cystic fibrosis in children. However, the authors conceded that it could not be ruled out that social, economic, or other factors determined both the smoking status of the household and the nutritional status of the children.

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Gilljam, et al., 1990	Reported that there was no statistical difference in clinical status or pulmonary function between children with cystic fibrosis from smoking and nonsmoking families. For patients with higher levels of physical activity, parental smoking appeared to matter less.
Young, et al., 1991	Reported that parental smoking may contribute to a higher level of airway responsiveness early in the child's life. The authors conceded that they did not report the actual amount of parental smoking due to the inaccuracies of parental reporting.

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PARENTAL SMOKING AND OTITIS MEDIA IN CHILDREN

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OTITIS MEDIA ("GLUE EAR")

In addition to claims that parental smoking affects the respiratory health of children, there are claims that exposure to parental smoking might increase the risk of otitis media (commonly known as "glue ear") in children. Otitis media is an inflammation of the inner ear that often leads to an accumulation of fluid in the inner ear canal. Otitis media often appears as a complication of various viral and bacterial infections, including measles. If left undetected, otitis media can lead to hearing loss and learning disabilities. Authors of studies examining this issue have not formulated a widely accepted mechanism for how ETS might increase the risk of otitis media in children. There are two theories that are proposed but have not been scientifically proven: 1) One theory is that ETS somehow chronically irritates the eustachian tube of the inner ear, leading to the inflammation; and 2) The second theory is that ETS potentially increases the risk of upper respiratory tract infections that spread to the inner ear. The results of the studies on this subject are variable and are subject to the same confounders that potentially bias the results of studies on other ETS related issues. While the science on this issue is controversial, the reported seriousness of otitis media makes investigators anxious to make recommendations for child safety. However, the studies on otitis media and parental smoking have not established a clear association.

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RESULTS OF SELECTED STUDIES ON OTITIS MEDIA

Said, et al., 1978	Reported that number of tonsillectomies/adenoidectomies were related to the amount of parental smoking in the home
Pukander, et al., 1985	Reported that parental smoking in the home increased the risk of acute otitis media and that breast-feeding seemed to have a protective effect
Kallail, et al., 1987	Reported that parental smoking was not associated with the occurrence of otitis media
Zielhuis, 1989	Reported that parental smoking was not related to an increased risk of otitis media
Strachan, 1990	Reported that parental smoking was an important determinant of middle-ear underpressure and effusion
Strachan, 1989	Reported that approximately one-third of the cases of middle ear effusion in their subjects were attributable to passive smoking
Taninio, et al., 1988	Reported that parental smoking was more prevalent among the subjects with recurrent otitis media
Kraemer et al, 1983	Reported that parental smoking, atopy, and catarrh posed the greatest risk to children of developing persistent middle-ear effusions when present together in the same child

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Pukander, et al., 1990	Reported that maternal smoking increased the risk of acute otitis media in infants and especially of recurrent attacks
Vinther, et al., 1982	Reported that there were no effects of parental smoking on the frequency of otitis media in children
Moorhead, 1985	Reported that otitis media was found to be related to parental smoking in this general practice study
Black, 1985	Reported that parental smoking increased a child's risk of undergoing surgery for otitis media
Fleming, et al., 1987	Reported that full time day-care attendance increased the risk of ear infections (odds ratio = 3.8)

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ETS AND ADULT RESPIRATORY DISEASE/SYMPTOMS

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ADULT RESPIRATORY DISEASE

Several studies have examined respiratory disease and symptoms and their possible association with exposure to environmental tobacco smoke in adults. The studies that examined respiratory disease and symptoms in adults are contained in this section. For additional studies on adults, see the "compromised individuals" and "lung function" sections.

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RESULTS OF SELECTED STUDIES: ETS AND ADULT RESPIRATORY HEALTH

Lee, 1986	Passive smoking was not associated with an increased risk of chronic bronchitis in the nonsmoker.
Koo, et al., 1988	Reported an association between respiratory symptoms in the mothers and the same symptoms in their children. Indicates that cross-infection is an important confounder of studies on parental smoking and childhood respiratory health.
Hole, et al., 1989	Reported an association between passive smoking and adverse cardiorespiratory symptoms in adult nonsmokers. None of the associations, however, were significant.
Koo, et al., 1990	With the exception of smoking by the father and the children's NO2 levels, no association was found between smoking at home and NO2 levels.

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ETS AND ADULT LUNG FUNCTION

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LUNG FUNCTION IN ADULTS

The studies that have investigated lung function in adults and its possible relationship to environmental tobacco smoke exposure are presented in this section. To aid in the interpretation of this literature, definitions of the major lung function parameters are provided below.

Df: One of the most widely used measures of pulmonary function in adults and children is forced vital capacity and is represented in the literature as FVC. This term refers to the maximum volume of gas that a person can expire as forcefully and rapidly as possible from their lungs immediately following a maximal inspiration of air. When a person's ability to expire air forcefully and rapidly from their lungs (FVC) is compromised, this can possibly be an indication of chronic obstructive lung disease. Decreased FVC is common in restrictive diseases such as pulmonary fibrosis and in obstructive diseases such as emphysema and asthma.

Df: A second important measure of pulmonary function is the forced expiratory volume in one second, which is abbreviated as FEV1 in the literature. The FEV1 measure is simply the amount of air that is expired in the first second of the FVC maneuver. As with FVC, this parameter is useful in the assessment of airway obstruction. The two parameters, FVC and FEV1, are often used in a ratio to determine the percentage of a person's FVC that is expired in the first second of the maneuver. A FEV1/FVC ratio lower than 65% to 70% is characteristic of obstructive lung disease. On the other hand, subjects with restrictive lung disease will often show a normal or exaggerated FEV1/FVC value.

Df: Forced expiratory flow, known as FEF25%-75%, is the average rate of flow of air during the middle half of an FEV maneuver. The FEF25%-75% is indicative of the status of the medium and small sized airways. Decreased values of FEF25%-75% are common in the early stages of obstructive lung disease. Low values of FEF25%-75% in combination with normal values of FVC and FEV1 are often indicative of early small airways abnormality. Reduced FEF25%-75% are sometimes seen in cases of severe restrictive disease as well.

All of these measures share a common problem: accurate assessment requires the full cooperation and maximal effort of the subjects under investigation. Accurate measures are sometimes

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therefore difficult to obtain. The studies are not consistent in the lung function parameters they measure, and there is also a lack of consistency among the results of the same function tests across studies. Following is a presentation of the major studies that have examined these lung function parameters in adults. The investigators who have found associations between impaired lung function and ETS exposure are often uncertain of the clinical meanings of the small decreases observed in their studies. Therefore, it is not suprising that no definitive conclusions have been reached regarding ETS exposure and its possible association with lung function in adults.

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REFERENCE

Ruppel, Gregg. Manual of pulmonary function testing (Fourth edition). The C.V. Mosby Company, 1986, pp. 33-38.

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RESULTS OF SELECTED STUDIES: ETS AND ADULT LUNG FUNCTION

Bouhuys, et al., 1978	The authors reported no associations between smoking in the home and increased symptoms or lung function loss among nonsmokers living in the same households.
Shephard, et al., 1979	The authors raise the possibility that subjective reporting of symptoms could have been "suggested" by the odor of the cigarette smoke.
White, et al., 1980	This study has received numerous criticisms.
Comstock, et al., 1981	Passive smoking in the home was not associated with the prevalence of respiratory symptoms and was only "suggestively associated" with impaired ventilatory function.
Kauffmann, et al., 1983	Opposite trends in FEV1 and FEF25-75 were found in men passively exposed to tobacco smoke, and the differences observed in women were slight and not statistically significant.
Jones, et al., 1983	The use of cooking fuels was found to be associated with impaired ventilatory function in a group of nonsmoking women.
Kentner, et al., 1984	Passive inhalation of tobacco smoke at home or the workplace was found not to be associated with impaired lung function in healthy nonsmokers.
Lebowitz, et al., 1985	Reported no direct association between ETS and lung function parameters in adult nonsmokers.

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Hosein, et al., 1986	The use of gas stoves was found to be associated with impaired lung function in women. It was reported that passive smoking in households where gas stoves were used appeared to have no effect on lung function values.
Masi, et al., 1988	The authors concede that their use of multiple tests of significance (involving both exposure and response measurements) are likely to have resulted in some associations achieving statistical significance by chance.
Kalandidi, et al., 1990	Assessment of exposure was based solely on the husband's smoking habit in terms of amount (daily), amount (total), and duration.

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ETS AND COMPROMISED ADULTS

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COMPROMISED ADULTS

The literature on environmental tobacco smoke includes a body of research on asthmatic adults. Studies have been conducted in order to attempt to determine whether there is a relationship between passive exposure to ETS and the development of asthma or the exacerbation of existing asthma in adults. The studies are varied in their results, and accordingly, no definitive conclusions have been reached by investigators. Following are the studies that examine ETS and its possible relation to asthma in adults.

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COMMENTS ON SELECTED STUDIES: ETS AND COMPROMISED ADULTS

Stahle, et al., 1978	The authors suggest that tobacco smoke exposure might trigger asthma attacks by means of an allergic reaction.
Shephard, et al., 1979	Reported that asthmatic subjects did not appear to have an unusual sensitivity to tobacco smoke exposure.
Dahms, et al., 1981	Five of the ten subjects specifically reported sensitivity to tobacco smoke before their inclusion in this study.
Ing, et al., 1983	This study investigated only six subjects.
Romer, et al., 1983	The authors concede that the small subject population of this study indicates that the results must be taken with caution.
Knight, et al., 1985	Only six subjects were studied.
Wiedemann, et al., 1986	The authors report that passive smoking presents no acute respiratory risk to young asymptomatic asthmatic patients.
Stankus, et al., 1988	Only 21 subjects were included and all had complained of respiratory symptoms upon previous exposure to environmental tobacco smoke.
Bailey, et al., 1990	No relationship was observed between passive smoking and pulmonary function of asthmatic subjects.

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CONFOUNDERS

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CONFOUNDERS

Studies on parental smoking and childhood respiratory disease rarely address confounding variables. Confounding variables are factors that can create a "false" association between two elements by being associated with one or both of them. For example, factor X (socioeconomic status) may be associated with both factor Y (parental smoking) and factor Z (childhood respiratory disease). When factor X is not controlled for in epidemiological studies of the possible association between factor Y and factor Z, a false association may appear between factors Y and Z. Therefore, it is vital that epidemiologists control for confounding variables when conducting studies such as those on parental smoking. The possible confounding variables associated with parental smoking and childhood respiratory disease can be grouped into four major categories: (1) household heating and cooking sources; (2) outdoor air pollution; (3) organic substances; and (4) demographic, medical and socioeconomic factors.

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Household heating and cooking sources

Children living in households with gas stoves have been reported to have a greater history of respiratory illness before the age of two and small but significantly lower levels of FEV_1 and FVC corrected for height[1] (FEV1 and FVC are standard measurements of lung capacity and function). Similarly, exposure of children to gas cooking in the first two years of life has been associated with an increased risk of hospitalization for respiratory illness[2]. There are reported associations of gas stove use with daily peak flow in asthmatic, normal, and allergic subjects.[3]

Oxides of nitrogen (NOx) arising from the use of gas stoves for cooking were proposed to be related to a reported increase in cough, "colds going to the chest," and bronchitis in a study of 5,758 English and Scottish children aged six to eleven years[4]. A number of other confounders were controlled for in this study, including "age, social class, latitude, population density, family size, overcrowding, outdoor levels of smoke and sulphur dioxide and types of fuel used for heating." One group of researchers reported similar results for a five-year longitudinal study of 4827 boys and girls, ages five to ten years. This reported association was independent of age, sex, social class, number of cigarette smokers in the home, and latitude, and was only found in urban areas.[5]

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Use of unvented kerosene heaters, which release nitrogen dioxide (NO_2) into the indoor environment, was associated with significantly more days of acute respiratory illness in exposed children[6]. In this study, there was no difference in the number of cigarettes smoked daily in the homes of exposed versus unexposed children. NO_2 exposure was also reported to be associated with a risk of reporting lower respiratory symptoms in children under the age of seven[7].

One study reported increased proportions of chest illnesses and hospitalizations for chest illness before age two in young children living in homes heated by wood-burning stoves. Medical histories, sociodemographic factors, or exposure to other pollutant sources did not account for the reported association[8].

In another report, hot water heating systems were reported to have a large effect on lung function in children, when compared to the use of forced air heating and air conditioning systems[9].

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Outdoor air pollution

Outdoor air pollutants have been identified as a confounder in several studies. In one study, acute respiratory disease incidence was reported to be positively associated with higher ambient sulfate levels[1].

A group of researchers examined the importance of indoor and outdoor environmental factors (parental smoking, gas cooking, suspended particulates and sulfur dioxide) in the respiratory health of seven- to ten-year-old Canadian children. The researchers were unable to identify any effects of parental smoking or gas cooking because the prevalence of these variables was highest in an industrial area of high particulate pollution[2].

One researcher has reported a strong association between respiratory illness and particulate pollution in children living in a study site which experiences relatively high levels of particulate pollution[3].

A study comparing Israeli children living in a polluted industrial town versus those living in an unpolluted area reported that chronic respiratory symptoms and most pulmonary diseases were significantly more common among those children from the polluted town[4].

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Organic substances

The relevance of home dampness in the etiology of respiratory symptoms in children is supported by current research which links dampness with the presence of molds, dust mites, fungi and other allergenic microbes.

In one study, odds ratios of 1.23 and 2.16 were reported for home dampness after adjustment for several factors, including maternal smoking, in a cohort study of 4,625 eight- to twelve-year-old children living in six United States cities[1]. The authors reported odds ratios for molds of 1.27 to 2.12 after adjustment for maternal smoking and several other factors.

Another study reported higher rates of respiratory symptoms and symptoms of infection and stress among children living in damp houses. The presence of "fungal mould" was also reported to be related to higher rates of respiratory symptoms, independent of smoking in the household[2]. In another study, the growth of fungi and molds in the home was directly related to respiratory symptoms and sensitization to common allergens in children[3].

Researchers have reported that children living in damp and moldy dwellings had a greater prevalence of respiratory symptoms and headache and fever than those living in dry homes. The authors reported a dose-response relationship with increasing

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numbers of symptoms reported in dwellings with higher severity of dampness and mold. All these differences persisted after controlling for possible confounding factors such as household income, cigarette smoking, unemployment, and overcrowding[4].

Atopic sensitization of children to house dust mites was reported to be related to home dampness[5].

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Demographic, medical and socioeconomic factors

Low socioeconomic status has been associated with an increased incidence of respiratory complications[1]. Factors related to lower socioeconomic status include: inadequate medical care, poor nutrition, poor outdoor air quality, increased parental coughing, higher gas stove usage, frequent change of address, and lower per capita living space. In a study of 1,050 European children aged eight and nine years, lifetime and current prevalence of wheeze were both significantly higher in children from low socioeconomic status[2].

Watkins, et al., (1986) reported high consultation rates for respiratory illness in children whose fathers were in manual occupations. This association was not explained by crowded home conditions or parental smoking[3]. Gardner, et al., (1984) reported significantly higher rates of lower respiratory disease in infants of low socioeconomic status[4].

Cross-infection also plays a role in the incidence of children's respiratory disease. For instance, in a 1988 paper, Koo, et al., reported that among Japanese and Hong Kong Chinese women, there was a highly significant correlation between the frequency of maternal respiratory illness and the frequency of respiratory illness in her children[5].

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Cross-infection may be relevant to the reports of associations between day care attendance and respiratory illness. Anderson, et al., (1988) reported that care outside the home (day care) is an important factor for acquiring lower respiratory tract illness and infectious diseases in children under two years of age[6]. Gardner, et al., (1984) also noted significantly higher rates of lower respiratory disease among day care infants[4]. Fleming, et al., (1987) reported an increased risk for upper respiratory tract infection associated with day care attendance[7].

Familial characteristics and genetics may also act as confounders. For instance, in a 1982 publication, Lebowitz, et al., report that an observed relationship between children's pulmonary function and parental smoking disappeared when household aggregation of body mass was taken into account[8]. Another Lebowitz, et al., study (1984) also reported that there was "no remaining independent aggregation of pulmonary function measurements" after familial aggregation of body habitus was controlled for[9]. Genetic predisposition may play a role in respiratory illness and pulmonary function[10]; although cross-infection is also involved[11].

"Lifestyle" may also act as a confounder. A study in Copenhagen (Holma and Winding, 1977) examined 109 social, medical, housing, and hygiene factors on morbidity. The best predictors for health were "thriving" (satisfaction), followed by "housing standard" and "personal hygiene." The authors reported no effect

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of parental cigarette smoking on the respiratory health of young children[12]. A survey of 314 nonsmoking Hong Kong Chinese women and their children and 243 Japanese women and their children reported that chronic cough and sputum symptoms were at least 10 times more prevalent in Hong Kong[5]. This observation was attributed to occupational exposure to dust or fumes and household crowding among the Hong Kong mothers.

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PARENTAL SMOKING: CONFOUNDING VARIABLES

Access to medical care

Age of mother

Air pollution

Birth weight

Breast feeding

Cooking practices/type

Day care attendance

Diet

Family history of illness

Family size

Gender of child

Genetic determinants

Heating type

Home dampness

Hospital spread of illness

Household pets

Newborn illnesses

Nurture

Overcrowding

Parental education

Parental infections

Place of residence

Seasonal variation

Skin test reactivity (allergy)

Socioeconomic status

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I. HEALTH CLAIMS

- The claim that exposure to environmental tobacco smoke (ETS) in the workplace causes disease in nonsmokers is not justified on a scientific basis.

Exposure

- Measurements taken in offices, workplaces and public places indicate that the contribution of tobacco smoke to indoor air is minimal.[1] For example, typical nicotine measurements (which are particularly revealing because nicotine is unique to tobacco smoke) range from an exposure equivalent of 1/100 to 1/1000 of one filter cigarette per hour.[2] In other words, a nonsmoker would have to spend from 100 to 1000 hours in an office, restaurant or public place in order to be exposed to the nicotine equivalent of just one cigarette.

Lung Function

- A 1980 report[3] which concluded that nonsmokers exposed to tobacco smoke at work for 20 or more years had reduced function of the small airways compared to nonsmokers not so exposed still receives considerable attention, although it was heavily criticized for questionable data acquisition and analysis.[4] In contrast, a more recent study of 1,351 German office workers reportedly found "no evidence" that everyday exposure to

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tobacco smoke in the office or at home leads to an essential reduction of lung function in healthy adults.[5]

Lung Cancer

- Nine of the published studies on spousal smoking and lung cancer examined workplace exposure to ETS and the incidence of lung cancer in nonsmokers.[6] Not one of the studies provides adequate support for an association between ETS exposure in the workplace and lung cancer.

Allergy

- One of the most widespread beliefs, especially in the workplace setting, is that some nonsmokers are "allergic" to tobacco smoke. Scientific researchers, however, have not identified specific allergens in tobacco smoke.[7] Thus, while some individuals may react to the sight or smell of tobacco smoke, this does not mean that they are experiencing an "allergic" reaction to it.

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II. SICK BUILDING SYNDROME

Because it is visible and easily identified by its aroma, environmental tobacco smoke is often blamed for indoor air quality problems. Government and private studies of "sick buildings" in the United States, Canada and Europe report, however, that tobacco smoke may be involved in only two percent to five percent of the buildings investigated for complaints about air quality.[1] For example, from 1981 through 1987, one company studied indoor air quality in 223 different buildings, accounting for over 39 million square feet of property, and found that ETS was a significant concern in only 10, or 4%, of the buildings [Robertson, 1988]. In another SBS database, smoking was implicated as a major contributor to complaints in only 12 of 408 (<3%) of the buildings surveyed [Collett, 1989]. The National Institute for Occupational Safety and Health (NIOSH) investigated more than 200 "sick" buildings and found that tobacco smoke was the source of claimed discomfort in only 2% of the buildings investigated. Ventilation problems were associated with half the complaints; outdoor air was considered a bigger problem than ETS [Melius, 1984]. Other investigators concluded that bacterial and fungal contamination is a major source of indoor air problems [Collett, 1989; Robertson, 1988].

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- Inadequate ventilation exacerbates all indoor air quality problems. This suggests that even a total smoking ban is not likely to affect comfort problems in 95 to 98 percent of "sick buildings."[2]

- The majority of indoor air quality problems in "sick buildings" have been traced to inadequate fresh air and poor air filtration. Because the visibility of tobacco smoke may be an indicator of inadequate ventilation, the prohibition of smoking serves to mask the real reason for poor indoor air quality--lack of proper ventilation. In addition, concentrating on tobacco smoke ignores the fact that adequate ventilation should always be provided in any enclosed space, regardless of whether or not smoking is permitted.

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III. VENTILATION

- In 1981, The American Society of Heating, Refrigeration and Air-Conditioning Engineers (ASHRAE) issued a ventilation standard for public places (ASHRAE 62-1981).[1] The Standard established two levels of ventilation, one for areas in which smoking was permitted, and another substantially lower rate for areas where smoking was prohibited. The Standard was recently revised and reissued (ASHRAE 62-1989) with one prescribed ventilation rate, regardless of whether smoking was permitted or not. The decision to reject separate ventilation rates for smoking and nonsmoking areas was influenced by two areas of research: (1) The amount of ventilation required to remove indoor contaminants produced by humans, namely carbon dioxide and body odor, is also sufficient to remove typical amounts of ETS; and (2) ventilation rates for nonsmoking areas under ASHRAE 62-1981 were found to be inadequate and permitted airborne substances to increase, even in the absence of ETS.[2]

- Operating costs for increasing ventilation to meet specifications set forth in ASHRAE 62-1989 have been estimated at 3-5% over current annual energy expenditures for office buildings. Compliance with ASHRAE Standard 62-1989 will apparently have little effect on annual energy budgets.[3]

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IV. ACCOMMODATION

- Since the claim that exposure to tobacco smoke causes disease in nonsmokers is not scientifically justified,[1] the real issue regarding the "right" to smoke-free air is whether or not smoking should be prohibited because some people consider it to be an annoyance or nuisance.

Annoyance:

- Tobacco smoke may be an annoyance or nuisance to some people, but such complaints typically arise in poorly ventilated areas. Such complaints are most frequently associated with inadequate ventilation and to indoor substances other than ETS [See Section II: "Sick-Buildings"].

- Regulating a behavior such as smoking merely because some see it as an annoyance has undesirable consequences. Numerous individual behaviors could fall into the category of "annoyances," and to demand restrictions on all those potentially "annoying" behaviors is "to call for government regulation of everything."[2]

- Such regulations also reject the real possibility that people can work things out among themselves and may place a minority

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of individuals in the position to dictate what is "right" for everyone.

- The alternative to intrusive regulation is good manners, common courtesy and cooperation between smokers and nonsmokers. This alternative preserves the delicate balance of individual rights and allows for accommodation of everyone's desires.

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The Separation Issue:

- The 1986 Report of the Surgeon General on ETS suggests that separation of smokers and nonsmokers is not effective in minimizing the nonsmoker's exposure to ETS. That claim was made without scientific support. Indeed, subsequent research indicates that simple separation of smokers and nonsmokers effectively minimizes ETS exposures for nonsmokers.[1-3]

- One recent study reported that the use of designated smoking areas reduced exposure to ETS by 95%.[1] Another study of a smoking-restricted office building reported that ambient nicotine in nonsmoking areas was virtually undetectable, suggesting that ETS had a negligible impact on the nonsmoking areas of the building.[2]

- Canadian researchers, in a series of reports, presented results on levels of ETS constituents in offices where smoking was regulated and unregulated. They reported no significant differences in average ETS constituent levels between nonsmoking offices that received recirculated air from designated smoking areas and nonsmoking offices that did not receive recirculated air.[3] They concluded:

> The results indicate that the provision of a designated, but not separately ventilated smoking area can effectively eliminate or

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drastically reduce most components of environmental tobacco smoke from nonsmoking offices.

- A federally-sponsored study of ETS in aircraft cabins indic that separation of smokers and nonsmokers is an effective me for reducing exposure to ETS.[4] The authors reported that m ETS constituent concentrations in nonsmoking sections w below the limit of detection. Similar results have b reported in other studies.[5]

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